Most of Dr. Birdwood's missionary zeal is harmless, and some of it is a useful reminder of our age-old obligations to the sick. But I must protest at his attack on health centres and at what is suspiciously like a sneer at the Royal College of General Practitioners' "ideal"; his experience of both must be lamentably small and/or distorted.

Health centres—of all shapes and sizes—are modern medical devices designed to enhance the delivery of primary, personal, continuous, comprehensive, family care. As one who has visited, and will continue to visit, many health centres in Britain, I can assert that, despite some unfortunate errors in design, the bringing to bear on every individual patient's problems of the whole range of caring professions and their relevant technologies holds better promise of making and keeping people well than does the traditional, fragmented, isolated system we have known until recently. I challenge Dr. Birdwood to produce evidence—real evidence—that the health centre concept reduces the personal element in care.

Any institution which tries to provide leadership, especially in the educational field, must expect to be criticized, and a good institution should welcome well-reasoned constructive criticism. To swipe, as Dr. Birdwood does, at a carefully thought out set of objectives (including group practice, team care, and health centres) published by the Royal College of General Practitioners, and to do it almost as an aside in a backward-looking article, is to my mind irresponsible. It has been my good fortune to meet, listen to, and argue with, many of the office-bearers of that College and such experience makes me confident that these men, and the doctors they represent, will never allow the humane element of general practice to suffer from a surfeit of technology.

Yes, Dr. Birdwood, let our profession show the right road—and that is forward into the new general practice where humanity uses technology and not backward to where the one smothers the other.—I am, etc.,

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Hypophosphataemia and Hypokalaemia

SIR,—We would like to add our observations on hypophosphataemia and its association with hypokalaemia to those of Dr. D. C. Anderson and colleagues (15 November, p. 402).

Seven out of fourteen patients treated by us for idiopathic hypercalciuria¹ have shown a persistent fall of over 1 mg./100 ml. in plasma phosphate immediately following commencement of therapy with bendrofluazide 5 mg. per day. In two patients bendrofluazide was stopped after two and four years' treatment and the low plasma phosphate rapidly returned to normal. Re-commencement of bendrofluazide again resulted in hypophosphataemia. In two patients treated for two and ten years, hypophosphataemia persisted when bendrofluazide was stopped. Potassium supplements given to the patient treated for ten years,

and to two other patients with "bendrofluazide induced" hypophosphataemia did not result in any improvement in plasma phosphate levels. In two patients with irreversible hypophosphataemia, renal tubular reabsorption of phosphate was assessed. The first had a tubular maximum reabsorption of phosphate of 90 μ moles per minute (normal 165 ± 25 μ moles per minute), and the second a T.m./G.F.R. of 24 mg./l. (very low normal) measured by the method of Bijvoet *et al.*²

Prolonged bendrofluazide therapy may, therefore, result in irreversible hypophosphataemia owing to defective renal tubular reabsorption of phosphate. Although plasma potassium levels never fell below 3.5 m. Eq/l. in any of our patients receiving bendrofluazides, it is known that the vast majority of individuals treated with thiazide diuretics have a low total body potassium.³ It is, therefore, possible that an intracellular potassium deficiency over a prolonged period of time may cause irreversible renal tubular damage and result in a renal phosphate leak. An alternative explanation is that thiazide diuretics may cause parathyroid overactivity which would account for defective renal tubular reabsorption of phosphate.⁴ Hypercalcaemia due to parathyroid adenoma or hyperplasia has been reported in eight patients receiving long term thiazide therapy.⁵ There was, however, no evidence of hypercalcaemia in any of our patients, and plasma ionized calcium (measured on two separate occasions at Professor C. G. Dent's Metabolic Unit, University College Hospital) in the patient with a low T.m.p. was normal.

The absence of bone disease in our patients is attributed to the fact that intestinal phosphate absorption is normal. In the two patients in whom renal tubular handling of phosphate was assessed, plasma phosphate rose from 1.5 and 1.8 mg. per 100 ml. to 3.8 and 3.2 mg. per 100 ml. respectively following an oral load of 6.9 g. of sodium phosphate dissolved in 200 ml. of water.—We are, etc.,

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Fluid Therapy in the Post-operative Period

SIR,—I read with interest your correspondent's report of the meeting of the International Society of Surgeons in Buenos Aires (18 October, p. 164), and I would be grateful if you would allow me to correct the following misconceptions.

I did not advocate the administration of one-fifth normal saline intravenously to elderly post-operative patients in sufficient quantities to produce a urinary output of 70 ml. each hour, nor did I advocate the maintenance of a venous pressure in such

patients of 5-20 mm. water. In a section dealing with post-operative fluid and electrolyte therapy in elderly patients I pointed out that in recent years, following the work of Shires and his colleagues,¹ there has been a tendency in all age groups to reverse the old regimen of salt and water restriction in the early post-operative period. In a symposium on post-operative geriatric care, Swartz² advocated maintenance of central venous pressure between 8 and 15 cm. of saline and urine output at 40 ml./hr. or more. He recommended that a Foley catheter and central venous pressure catheter should be inserted. Intravenous orders are then instituted as follows: "Replace previous 1 hour urine volume plus 30 ml. with one-third normal saline in 5% dextrose and water." If urine output remains less than 40 ml./hr. for 2 hours and central venous pressure is less than 20 cm. of saline then 25 g. of mannitol would be infused rapidly. If urinary output increased the same programme of hourly fluid replacement would continue.

However, even in patients of a younger age group reports appear from time to time of congestive failure and pulmonary oedema.³ Furthermore, Roth, Lax, and Maloney³ have recently refuted the evidence of Shires and his colleagues.

In common with Swartz we feel that bladder catheterization is justified in elderly patients likely to develop problems with post-operative fluid balance, despite the danger of infection, and central venous pressure and hourly urine output are also monitored. We differ from Swartz, however, in that we are considerably more cautious in the administration of salt in the early post-operative period to geriatric patients, and fluid is administered as 5% glucose or one-fifth normal saline in 4% glucose. Furthermore, though in our experience the range of normal central venous pressure is wide we are, in general, reluctant to accept a level above 10 cm. of saline in elderly post-operative patients.—I am, etc.,

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Unnecessary X-rays?

SIR,—As diagnostic radiologists we would like to support the views put by Dr. R. M. Scott on the necessity for "routine" radiographic examination of patients who have been involved in an accident (13 December, p. 689). While no one would dispute that the responsibility lies with the clinician it is also obvious that a very large number of x-ray examinations are requested for purely medico-legal reasons, particularly by the young and relatively inexperienced casualty officer to "cover" himself.

We do not criticize this attitude, but it is perhaps time that the legal aspects be examined by the medical profession, and that some eminent medical authority might take a stand against the present tendency to regard non-use of x-rays as almost amounting to malpractice. This is perhaps